MATERNAL AND PERINATAL OUT COME

In

MILD, SEVERE PRE ECLAMPSIA

AND ECLAMPSIA

A Comparative Study

M.D (Obstetrics & Gynaecology)

Part II

Department of Obstetrics & Gynaecology
Kilpauk Medical College
Chennai – 600 010.

TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY

September – 2006

BONAFIDE CERTIFICATE

This is to certify that maternal and perinatal out come in mild, severe preeclampsia and Eclampsia is the record work done by Dr. C. SivaKumar during his postgraduate course MD (2003-2006) in the Department of Obstetrics and Gynaecology, Kilpauk Medical college in partial fulfillment of the requirements for the award of the degree MD in obstetrics and Gynaecology as per the regulations laid down by Tamil Nadu Dr. M.G.R Medical University.

Prof.Dr.THIAGAVALLI KIRUBAKARAN,MD **The Dean** **Kilpauk Medical College** **Chennai.**	**Prof.Dr.INDRANI PADMANABHAN MD,DGO** **Head of the Department** **Department of obstetrics and Gynaecology,** **Kilpauk Medical College,** **Chennai.**

ACKNOWLEDGEMENT

I sincerely thank **Prof. Dr. THIAGAVALLI KIRUBAKARAN MD,** Dean, Kilpauk Medical College, Chennai for granting me permission to do this study.

I express my sincere thanks to **Prof. Dr. INDRANI PADMANABHAN M.D., DGO.** Head of the Department, Department of obstetrics and Gynaecology, Kilpauk Medical College Chennai.

I thank my PG teacher **Prof. Dr. NAGASUNDARI MD. DGO.,** Department of obstetrics and Gynaecology, Kilpauk Medical College, Chennai for her incredible guidance.

I thank **Prof. Dr. SRIDEVI M.D, DGO,** Registrar, Department of obstetrics and Gynaecology, Kilpauk Medical College, Chennai.

I extend my gratitude to **Mr. PADMANABAN, ICMR,** Kilpauk Medical College & Hospital, Chennai, for helpful guidance in statistics.

I am Thankful to all my professors, Assistant Professors and my colleagues for their support. Above all, I am thankful to all the patients in the study.

CONTENTS

INTRODUCTION

Pregnancy –induced hypertensive disease (PIH), in particular preeclampsia/eclampsia, remain important causes of maternal mortality and morbidity worldwide. The prevalence of PIH was 43.1 per 1000 pregnancies in a retrospective population-based study of 200,000 births in North Carolina, USA (Savitz 1992). For the past ten years, PIH has been the major cause of maternal mortality in England and Wales, and 56 per cent of deaths due to PIH were attributable to eclampsia (DOH report on confidential enquiries into maternal deaths in the United Kingdom 1985-1987). The Philippine Obstetrical and Gynecological Society committee on nationwide Statistics reported that 18.42 per cent of maternal deaths were due to PIH (POGS 1994).

PIH also contributes to a great extent towards fetal wastage as per latest studies it is responsible for 20% perinatal deaths and 2-3% maternal deaths.

PIH is defined as systolic blood pressure more than 140 mm of H_g or diastolic pressure more than 90mm H_g on 2 occasions 6 hrs apart in a previously normotensive pregnant women and associated with oedema and proteinuria.

The criteria for diagnosing PIH are onset of hypertension for the first time after 20 weeks of pregnancy and patients becoming normotentive after delivery.

This random study was taken up to evaluate the present status of maternal and perinatal outcome in mild and severe pre eclampsia and eclampsia in comparison with 200 normotensive control group at Kilpauk Medical College Hospital , Chennai in a period of 2003 to 2005.

AIM OF STUDY

1. To study the maternal and perinatal outcome in mild and severe pre Eclampsia and Eclampsia in comparison to the outcome in normal gravidae between the period of 2003 to 2005 at government K.M.C. Hospital, Chennai.

2. To study the factors that influence the maternal and perinatal outcome in mild, severe pre Eclampsia and Eclampsia.

3. To identify the factors affecting the maternal and perinatal outcome adversely so as to take steps to modify those factors for the improvement of materno fetal outcome.

4. To show the importance of proper antenatal, intranatal & postnatal care to improve the outcome.

REVIEW OF LITERATURE

The hypertensive disorders in pregnancy comprise several disease and Syndromes associated with high blood pressure. The disorders were called Toxemias of Pregnancy, but that term is an ambiguous misnomer.

Hypertension may antedate conception or arise during Gestation, or in the early puerperium. It is often accompanied by Oedema and Proteinuria separately or together and occasionally it culminates in convulsions and coma. The combined prevalence and incidence of the hypertensive disorders in pregnancy is probably about 6 percent, although vide variations have been alleged in relation to geographic, racial and socio economics factors.

The hypertensive disorders pose a serious threat to the Fetus and to the new- born infant who is often delivered prematurely either following spontaneous labour or by therapeutic termination of pregnancy.

MacGillvray in 1958 has found from his study that preeclampsia is primarily a disorder of first pregnancy **(MacGillvray 1958. Some observations on the incidence of pre-eclampsia - Journal of obstetrics and Gynaecology of British Commonwealth 65:**

The current hypothesis of Preeclampsia is a disorder of maternal and fetal incompatibility. This is consistent with the knowledge that Preeclampsia is more likely when there is an usually large trophoblast mass as with vesicular pregnancy, placental hydrops and multiple pregnancies and shown by **Chung et al., 1964 (Clinical Observations on some aspects of hydratiform mole journal of obstetrics and gynaecology British Commonwealth 71) Jeffcoate and Scott 1959 - some observation of placental factor in pregnancy toxemia American journal of obstetrics Gynaecology 72).** In these circumstances it is postulated that immune regulatory mechanism of normal pregnancy are overwhelmed by the size of antigenic load of placenta, particularly as moles as androgenic in origin and this fact is reassured by Kojil and Ohamma 1977.

CLASSIFICATION OF PREGNANCY INDUCED HYPERTENSION

ISSHP classification

Hypertension

Diastolic BP of ≥110 mmHg on any one occasion

(or) Diastolic BP of ≥ 90 mmHg on any two or more consecutive occasions ≥4 in apart.

Severe hypertension

Diastolic BP ≥120 mm Hg on any one occasion

(or) Diastolic Bp ≥110mm Hg on two or more consecutive occasion ≥4h apart.

Proteinuria

One 24h urine collection with a total protein excretion of ≥300 mg/24h.

(or) Two midstream or catheter specimens of urine (collected ≥4h apart) with ≥++ protein on reagent strip testing

(or) 3 + protein (if urine SG <1.030 and pH ≤8).

Recurrent Preeclampsia is diagnosed when hypertensive disorders occur in two or more pregnancies, not necessarily consecutive, with return of the blood pressure to normal after each delivery.

Pregnancy seems to be a screening test for later Hypertension. Gestational hypertension portends a high likelihood of late Chronic Hypertension.

MINIMUM CRITERIA OF DEFINING PREECLAMPSIA

Preeclampsia is a clinical diagnosis encompassing three elements (committee on obstetric hypertension in pregnancy ACOG 1996)

1. New onset hypertension (defined according to the latest American college of obstetricians and gynecology bulletin simply as a blood pressure consistently more than 140/90 mm Hg in previously normotensive women).
2. New onset Proteinuria defined as more than 300mg/24hrs or≥2+ on a clean catch dipstick in the absence of urinary infection)
3. New onset significant independent edema.

The diagnosis of PREECLAMPSIA should be made only after 20 weeks gestation

In the past it has been recommended that an increment of 30 mmHg systole or 15 mmHg diastolic blood pressure be used as a diagnostic criterion, even when absolute values were below 140/90 mmHg. This criterion is no longer recommended because evidence shows that women in this group are not likely to suffer (Levine 2000, North & Colleagues1999 (24) but warrant close observation. Conventional mercury sphygmomanometer remains the gold standard for blood pressure measurement. Blood pressure should be measured

with the women seated or at 45^0 reclined, with her feet supported or on the ground, and her arm at the level of the heart. The right arm should be used with the cuff of appropriate size. Electronic blood pressure monitors may underestimate the true pressure.

Nowadays it has been recommended that kortkoff phase V be used as a measure of diastolic pressure. (Brown et al 1998).

1. K4/K5 difference is smaller in hypertensive than in normotensive pregnant women.
2. K5 is closer to the actual intra-arterial pressure, physiologically more accurate, is more reliably detected and is reproducible.
3. K4 has limited reproducibility (shennam et al 1996)

PREECLAMPSIA is classified as either "mild" or "severe". There is no category of moderate preeclampsia.

SEVERE PREECLAMPSIA (ACOG)

A diagnosis of severe Preeclampsia should be entertained in women with new onset proteinuric hypertension and one or more of the following complications.

I. Severe elevations of blood pressure ≥ 160/110 mmHg on 2 occasions at least 6 hours apart.

II. Proteinuria (>5g/24h)

III. Oliguria <500cc/24hrs

IV. Cerebral, visual symptoms like blurred vision, scotomata, altered mental status, severe headache.

V. Symptoms of liver capsule distention (right upper quadrant or epigastric pain)

VI. Pulmonary Edema or cyanosis

VII. Thrombocytopenia (<100000 platelets / mm^3)

VIII. Hepatocellular injury (serum transaminase level > 2 times normal)

IX. Fetal growth restriction

THEORIES ABOUT CAUSES OF PREECLAMPSIA

1. Immunological mechanism - BARDEQUEZ(3)

There is immunological resistance to invading trophoblast by maternal immune system. Blocking antibodies and T helper cells, interleukins, interferon, growth factors play a major role. This results in inadequate trophoblast invasion of myometrial spiral arterioles.

2. Genetic predisposition - CHESLEY & COOPER 1986(6)

Susceptibility is by both single gene and multifactorial inheritance.

a. Women with angiotensin gene variant T232 had increased incidence

b. There is higher incidence of factor V Leiden mutation in Preeclamptic patients

3. Increased pressor response to angiotensinogen II - ABDUL KAREEM 1961 (1)

4. Altered vasoactive factors : VOLHARDT 1918 (45)

a. Endothelin - 1. A potent vasoconstrictor produced by endothelium is increased.

b. NITRIC OXIDE: A potent vasodilator is decreased. (Chang et al (5)

c. Reversal of PGI2 to TXA2 and vit.E ratio (Wang et al 1991 Walsh 1985(41)

d. Vasoactive maternal factor (VMF) has been imposed to cause the endothelial changes involved in the patho physiology of PIH.

5. Oxidants and Antioxidants

Hubal et al (18) have confirmed that Preeclampsia may have its origin in a disturbed oxidation mechanism. Under normal conditions equilibrium is maintained by anti oxidants. With increase in severity of Preeclampsia there is increase in lipid peroxidase and reduction in anti oxidants. Vit E lipid peroxidase cause endothelial damage.

6. Endothelial dysfunction : HAYMAN & ASS 2000(17)

Deficiency in trophoblastic invasion of placental blood spiral arteries leads to poorly perfused fetoplacental unit. This results in secretion of plasminogen activator inhibitor, into the maternal circulation leading to activation of endothelial cells to promote coagulation and increased sensitivity to vasopressor agents.

Banker and Coll 1995 have shown that BEGF levels are increased in serum of PE which may activate endothelial cells and release of inflammatory substances. (Dekar & Sibai 1998(8)

7. **Placental proteins**

Corticotrophin releasing factor, HCG, Activin A, Inhibin A are said to play a role.

8. Dietary deficiency: DAWSON, KELLY & Coauthors

Mac Gillivray viewed the evidence for a role of dietary deficiency in pathology of PE. It was concluded that when concentration of calcium is low in extra cellular fluid, amount of ionic calcium entering cell wall increases making vascular smooth muscle more sensitive to excitation.

9. Hyperhomocystinemia ; COLLER ET AL

Presence of infarcts retroplacentally is said to be cause of elevated levels of circulatory homocysteine. This is due to atherosis formed at placental site.

Elevated levels damage endothelium by H202 generation depletes nitric oxide mediated detoxification of homocysteine. Elevated levels of factor V increase in prothrombin activation.

PATHOPHYSIOLOGY OF PREECLAMPSIA

PRIMARY LEVEL

Changes that occur in placenta and placental vascular bed are the two lesions that involve spiral arterioles which are the end arteries supplying intravascular space

(REDEMAN 1991 (32)

a. Relative lack of trophoblastic infiltration of arterial walls during placentation **(Bloen et al 1972)**
b. Acute atherosis **(Robertson et al 1963(33)**
c. Magnitude of defective trophoblast invasion of spiral arterioles correlated with severity of hypertensive disorder.
d. Lipid accumulates first in myometrial cells and then in macrophages **(Madzli & Collegues 2000(20))**

SECONDARY LEVEL

1. Renal system

a. Decreased uric acid clearance, decreased glomerular function, decreased renal blood flow, Proteinuria. Intrinsic renal changes cause by severe vasospasm
b. Renal pathology : Proteinuria reflects advanced disease associated with poor prognosis **(Naeye & Friedman 1979)** Glomerular endotheliosis **(Sargo et Al 1959)**
c. Urinary sediments reflects renal changes **(Leduc et Al(19))**
d. Renal clearance of urate decreased **(Chesley & Williams 1945)**
e. Hypocalciuria **(Taufield et Al 1987(40)** because of increased tubular reabsorbtion
f. Hyperuricemia **(Pollok et Al 1960)**

g. Proteinuria **(Meyer & Colleagues 1994)** Trace or negative Proteinuria had negative predictive value only 34% in hypertensive women 3+ or 4+ Proteinuria were positively predictive of severe preeclampsia in only 36% of cases

II. **Cardio vascular system**:

a. Hemodynamic changes:

Increased arterial sensitivity to angiotensin II

Increased peripheral resistance

Decreased cardiac output

Increased BP

b. Blood volume

Hemo concentration, Blood volume decreased in women with homozygous for T 234 angiotensin genotype associated with preeclampsia **(Silver & Associates 2001(35).**

III. Coagulation system

a. Intravascular coagulation and less often erythrocyte destruction commonly in PE and especially Eclampsia **(Baker & Cunningham 1999)**

b. PT, aPTT, plasma fibrinogen level are unnecessary in management of hypertensive disorder of pregnancy **(Baker & Colleagues 1999)**

c. Thrombin time somewhat prolonged in a third of cases

d. Thrombocytopenia results from platelet activation, consumption and increased platelet production. Platelet aggregation is decreased compared with the normal increase seen in pregnancy **(Baker & Counningham 1999)**

e. Fragmentation hemolysis: **(Sanchel Ramos 1994(34))** Increased RBC fluidity in women with HELLP

f. Antithrombin III lowered **(Chang & Coworkers 1992(5))**

g. Fibronectin elevated **(Bru Baker & Colleagues 1992)**

h. Thrombophilias : Clotting factors deficiencies or mutation associated wuth early onset of preeclampsia

IV.Hepatic System:

a. With severe preeclampsia there is elevation of liver enzymes **(Combes & Adams 1972)**

b. Increased hepatic artery resistance by Doppler sonography **(Ooster Hof & Coworkers9(28))**

c. Periportal hemorrhagic necrosis in the periphery of liver **(Barton & Colleagues)**

d. HELLP syndrome : Hemolysis, elevated liver enzymes, low platelet **(Deboer & Coworker 1991(9)), Pritchard and Associates 1954(30), Weinstein 1985(43))**

IV. Endocrine system

a. Increased plasma levels of renin

b. Angiotensin II and aldosterone are decreased **(Weir & Colleagues)**

c. Arial natriuretic peptide is increased in women with preeclampsia **(Cunningham & Lindheimer 1999(7))**

V. Fluid and Electrolyte System

a. Volume of extra cellular fluid manifest as edema in women with severe preeclampsia has expanded beyond

the normally increased volume that characterizes pregnancy

b. Electrolyte concentration do not differ appreciably in PIH women compared with those of normal pregnancy.

TERTIARY LEVEL

Tertiary systemic effects of preeclampsia secondary to decompensation which presents as one of the following features

1. Eclampsia
2. Laryngeal edema
3. Cerebral hemorrhage
4. DIC
5. Corneal edema
6. Hepatic rupture
7. HELLP Syndrome
8. Retinal detachment
9. Renal cortical necrosis
10. Pulmonary edema
11. ARDS

PREDISPOSING FACTORS OF PREECLAMPSIA

1. Age

Young primi < 20 years

All patients > 30 years **Bobrowski & Bottoms 1995**

Showed increased incidence of HT & PIH > 35 years

2. Parity

Primi have incidence of 11.9% and multi have 4.7% Clinical Obs and Gyn. The incidence is 24$ new paternity multipara because of shorter period of Sperm exposure preceding conception.

3. Race

Davis in 1970 found an increased incidence in Muslims, Jews & Arabs. Africoamerican ethnicity is quoted to have increased incidence by Sibai 1997, Walker 2000

4. Social Status

Women of low social economic status are reported to have greater incidence of PIH, PE, Eclampsia. But Duffer & Mac Gillivray - 1968 found that difference between social classes are small if allowances are made for age, parity and levels of antenatal and intrapartum care. Baird and colleagues 1969 said that incidence was not different among five socioeconomic statuses.

5. Previous H/O Preeclampsia

The risk of preeclampsia in subsequent pregnancies is higher when it is

a. severe earlier and associated with low birth weight
b. Risk increases on increasing maternal age and interval between pregnancies.

Sibai et al studied subsequent pregnancy outcome in women with severe PE in first pregnancy

Risk of developing Eclampsia - 1.3%

Preeclampsia - 4.5%

Perinatal mortality - 5.9%

Norwegian study showed risk of 13.1% of PE in second pregnancy.

6. Family history

Severe preeclampsia, Eclampsia has a familial tendency. There is three fold increase of preeclampsia and four fold increase of severe PE. Chesley et al (6) found 26% incidence of PE in daughters. Lie et al found odds ratio 2.23 in sisters.

7. Pregnancy associated : Early onset

a. Twin gestation: Four fold increased risk Incidence of PE 25.3%. This is due to hyperplacentosis with increased placental hormone secretion relative placental ischemia or immunological reaction to the large placental mass

b. Molar pregnancy: confined to large rapidly growing moles in which the incidence of PE is 70%

(Page 1939(29)). With small slowly growing moles there is no increased incidence of PE.

c. Hydrops fetalis: Increased incidence due to hyperplacentosis.

d. Congenital malformations: In pregnancies complicated by triploidy, the risk of developing PE or hypertension in second trimester is 35% due to placentomegaly.

8. Urinary tract Infection

UTI resulting in an increased production of inflammatory products, cytokines, free radical species and proteolytic enzymes causes endothelial dysfunction **(Schieve et al)**

9. Underlying Disorders

a. Chronic hypertension is encountered in approximately 30-40% of early onset severe PE.

b. Underlying renal disorder : 20% have superimposed PE

c. Obesity, insulin resistance & Diabetes stone et al - BMI is an independent risk of severe PE

i. Obesity correlated with hypertension by expanded blood volume, Cardiac output to meet the increased metabolic demands.

ii. Obesity - dyslipidemia - delivery of free fatty acids to tissue, higher cholesterol / TG ratio, insulin resistance & hyperinsulinemia.

iii. Adipocytes release TNF alpha thereby involved in aggravating cytokine mediated oxidative stress.

iv. Insulin resistance / hyperinsulinemia associated with increased sympathetic activity and/or increased tubular sodium reabsorbtion thereby producing direct Hemodynamic changes.

v. Overt DM - 30% PE especially when vascular changes present. Khan & Daya showed odds of having PE increased by 20% per nmol/L increase in plasma glucose level.

10. Smoking

Decreases incidence of PE since it causes a significant reduction in HCG and estradiol level due to direct effect on the placental function **(Bernstein et al 1989)**

Management Objectives in severe preeclampsia

There is no preventive therapy against preeclampsia available at present even though aspirin, calcium magnesium, fish oil have been tried. Severe preeclampsia is associated with maternal complications like HELLP, Eclampsia, cerebral manifestations, abruption, fetal complications like IUGR, IUD, preterm delivery. As termination of pregnancy remains the only cure, the primary objective in the management of severe preeclampsia is to effect timely delivery in order to

- Prevent maternal morbidity and mortality
- Deliver a baby in an optimal condition, thereby minimizing perinatal morbidity and mortality.

In all circumstances, the well being of the mother is primary. In some circumstances, delays seriously jeopardize the well being of the mother, fetus or both.

MATERIALS AND METHODS

200 Cases of pregnancy induced hypertension complicating pregnancy admitted with duration of gestation between 21 weeks to 40 weeks were studied, 200 cases of normotensive patients of same period of gestation were taken as control group.

Group A- Normotensive control patients.

Group B- Pregnancy induced hypertension patients.

Apart from routine clinical examination the following aspects were studied in the mother and fetus.

I. STUDY OF MOTHER

Age, gravidity , parity, socioeconomic status, booking status, previous obstetric history, family history of hypertension, chronic nephritis, past history of hypertension, chronic nephritis, complications during pregnancy like anemia, Hypoproteinemia, APH, PPH, malpresentation malposition, urinary tract infection, diabetics mellitus, multiple pregnancy, abnormal liver function tests, with (or) without HELLP syndrome, fever, aspiration, DIC, renal failure, external injury, PROM , PPROM were noted.

Investigations like blood urea, sugar, serum creatinine, serum proteins, uric acid, electrolytes, serum fibrinogen, liver function tests including SGOT (AST), SGPT (ALT), Alkaline Phosphatase, prothrombin time, 24hrs urinary protein, specific gravity were noted.

On general examination distribution of Oedema was noted. Height, weight of patients recorded. Optic Fundus examined periodically for the presence of vascular changes. BP Checked 2 hrly & maximum pressure recorded since admission were noted. BP rechecked 24hrs 48 hrs, 1 week after delivery and on every visit up to 6 weeks.

Drugs like sedatives anti hypertensive & anticonvulsant were noted.

Follow up advice given to the patient for visit once in 2weeks up to 6 weeks.

II. STUDY OF FETUS

Apgar at one minute after birth was noted. sex incidence, weight of the babies at the time of delivery, maturity of the baby, perinatal mortality, sex incidence of fetal loss, ultrasonographic evaluation at different gestational age were noted . These babies were followed in the neonatal period few babies were followed up to 6 months.

RESULTS & OBSERVATION

Table - 1

Age Distribution in Normotensive and Hypertension

Complicating pregnancy

Age in Years	Normotensive Patients		PIH (mild + Severe + Eclampsia)	
	No. of Patients	%	No. of Patients	%
≤ 20 years	60	30	63	31.5
21-25	89	44.5	84	42
26-30 yrs	30	15	33	16.5
31-35 yrs	15	7.5	13	6.5
36 yrs and above	6	3	7	3.5
Total No. of Patients	200	100	200	100

P =0.96522563 X^2 =0.58 Statistically NOT SIGNIFICANT

In normotensive control patients maximum (44.5%) of patients belong to 21 to 25yrs of age, next 30% belong to ≤ 20 yrs of age, 15% belong to 26 to 30 yrs of age, 7.5% belong to 31 to 35 yrs of age, least 3% are 36yrs and above. Almost the same situation prevailing in PIH patients also. Maximum 42% of patients belong to 21 to 25 yrs age group. Next 31.5% belong to less than 20 yrs of age, 16.5% belong to 26 to 30 yrs of age, 6.5% belong to 31 to 35 yrs of age, least 3.5% of patients are 36 years and above.

Table - 2

Gravidity in normal and PIH Patients

Gravida	Normotensive (Control pts 200)		Mild PIH 92 patients		Severe PIH 68 patients		Eclampsia 40 patients	
	No. of Patients	%	No. of Patients	%	No. of Patients	%	No. of Patients	%
G_1 (Primi)	96	48	58	29	36	18	26	13
G_2	70	35	24	12	22	11	11	5.5
G_3	30	15	7	3.5	8	4	3	1.5
G_4 and Above	4	2	3	1.5	2	1	Nil	Nil

P =0.06851916 X^2 =7.11 statistically NOT SIGNIFICANT

In normotensive patients (total 200) 96 patients (48%) were primi Gravida. 70 patients (35%) were second Gravida. 30 patients (15%) were Gravida III and 4 patients (2%) were Gravida IV and above.

In mild preeclampsia, in a total of 92 patients 58 patients (29%) were primi Gravida 24 patients (12%) were second Gravida 7 patients (3.5%) were third Gravida. 3 patients (1.5%) were Gravida 4 and above.

In severe preeclampsia patients (total 68 patients) 36 (18%) were primi Gravida, 22 patient (11%) were second Gravida, 8 patients (4%) were III Gravida. Where as Gravida 4 and above are 2 patients (1%). In eclamptic patients (total 40) 26 patients (13%) were primi Gravida, 11 patients (5.5%) were Gravida II, 3 patients (1.5%) were III Gravida, Gravida IV and above were not present & there were no elderly primi Gravida.

TABLE -3

PARITY DISTRIBUTION

Gravida	Normotensive (Control pts 200)		Mild PIH 92 patients		Severe PIH 68 patients		Eclampsia 40 pts	
	No. of Patients	%	No. of Patients	%	No. of Patients	%	No. of Patients	%
Nullipara (Para `o')	96	48	58	29	36	18	26	13
Para I	76	38	28	14	25	12.5	12	6
Para II	26	13	4	2	5	25	2	1
Para III & above	2	1	2	1	2	1	Nil	Nil

P =0.01638577 X^2 = 10.27 Statistically SIGNIFICANT.

The parity distribution in normotensive group is as follows.

96 cases (48%) were Nullipara (Para 0), 76 cases (38%) were Para I, 26 patients (13%) were Para II, 2 patients (1%) were Para III and above. In mild preeclampsia (total 92), 58 (29%) cases were Para 0 (Nullipara), 28 (14%) cases were Para I and 4 patients (2%) were Para II, 2 cases (1%) were Para III and above.

In severe PIH (total 68 patients) 36 cases (18%) were Para 0**,** 25 patients (12.5%) were Para I and 5 patients (2.5%) were Para II, 2 patients (1%) were Para III and above.

In Eclampsia (total 40 cases), 26 (13%) cases were Para 0, 12 cases (6%) were Para I, 2 cases (1%) were Para II, no patients were above Para II.

Table - 4

BOOKING STATUS

Booking Status	Normotensive Control pts 200 cases		Mild PIH 92 patients		Severe PIH 68 cases		Eclampsia 40 cases	
	No. of Patients	%	No. of Patients	%	No. of Patients	%	No. of Patients	%
Unbooked patients	78	39	70	35	52	26	34	17
Booked	122	61	22	11	16	8	6	3

P =0.000001 X^2 =28.53 Statistically SIGNIFICANT.

In a total of 200 normotensive control patients 78 (39%) were Unbooked, 122 (61%) were booked.

In a total of 92 mild PIH cases 70 (35%) were unbooked, 22 case (11%) were booked.

In severe PIH (total 68 cases) 52 (26%) were unbooked, 16 cases (8%) were booked. In Eclampsia (40cases), 34 cases (17%) were unbooked, 6 patients (3%) were booked.

Table - 5

Socio economic status in normotensive and hypertensive patients

Socio Economic class	Normotensive 200 patients		Mild PIH 92 patients		Severe PIH 68 patients		Eclampsia 40 pts	
	No. of Patients	%	No. of Patients	%	No. of Patients	%	No. of Patients	%
Class I	Nil	-	Nil	-	Nil	-	Nil	-
Class I	Nil	-	Nil	-	Nil	-	Nil	-
Class III	22	11	3	1.5	1	0.5	Nil	-
Class IV	52	26	29	14.5	18	9	7	3.5
Class V	126	63	60	30	49	24.5	33	16.5

In 200 normotensive (control) patients, no one belongs to class I (or) class II socioeconomic status. 22 cases (11%) belong to class III, 52 (26%) were in class IV and majority 126 cases (63%) were in lowest class V socioeconomic status. In 92 cases of mild PIH none belongs to class I, II. Only 3 patients (1.5%) belong to class III, 29 (14.5%) cases were class IV, 60 patients (30%) were class.

In severe PIH patients (68 cases) Class I, II cases were NIL, only 1 patient (0.5%) is class III 18(9%) were class IV, and 49 cases (24.5%) were class V. In eclamptic patients (total 40) none belongs to class I, II and III. 7(3.5%) patients belong to class IV and majority 33 patients (16.5%) were from lowest socio economic class V.

Table - 6

Previous BOH in normotensive and Hypertensive patients

Previous history	Normotensive 200 patients		Mild PIH 92 patients		Severe PIH 68 patients		Eclampsia 40 pts	
	No. of Patients	%	No. of Patients	%	No. of Patients	%	No. of Patients	%
Previous IUD	Nil	-	Nil	-	1	0.5	Nil	-
Previous abortions	8	4	6	3	3	1.5	1	0.5

In normotensive controls past obstetric history reveals 8 (4%) patients had previous abortions. There is no previous IUD in this group.

In PIH patients 1 patient (0.5%) gave the history of previous IUD at 34 weeks after a bout of high fever for 3 days. There was history of previous abortions of about 10 (5%).

Table - 7

Past and family history of hypertension (Mother, Father I degree relative)

Group	History of Hypertension in the previous pregnancy		History of Hypertension in the family	
	No. of Patients	%	No. of Patients	%
Normotensive	Nil	-	14	7
PIH	29	14.5	19	9.5

In normotensive control no one gave the history of hypertension in the previous pregnancy) but 14 cases (7%) gave history of hypertensive disorder in the family members (father, mother, sibling).

In PIH patients 29 cases (14.5%) gave the history of hypertension in the previous pregnancy (recurrent PIH), 19 cases (9.5%) gave the history of hypertension in the family.

Table - 8
Complication during present pregnancy

Nature of Complication	Normotensive (Control pts 200)		Mild PIH 92 patients		Severe PIH 68 patients		Eclampsia		Total percentage in PIH patients
	No. of Patients	%	No. of Patients	%	No. of Patients	%	No. of Patients	%	
Anemia	45	22.5	14	7	9	4.5	25	12.5	24
Hypoproteinemia	7	3.5	6	3	9	4.5	24	12	19.5
APH	Nil	-	-	-	3	1.5	1	0.5	2
PPH	1	0.5	-	-	1	0.5	1	0.5	1
Mal presentation	7	3.5	5	2.5	2	1	2	1	4.5
Mal Position	5	2.5	3	1.5	2	1	3	1.5	4
UTI	Nil	-	6	3	18	9	23	11.5	23.5
Diabetes Mellitus	Nil	-	4	2	2	1	1	0.5	3.5
Multiple Pregnancy	1	0.5	Nil	-	Nil	-	Nil	Nil	Nil
Abnormal Liver Function test	Nil	-	Nil	-	43	21.5	37	18.5	40
HELLP (DIC)	Nil	-	Nil	-	Nil	-	3	1.5	1.5
Fever	Nil	-	5	2.5	7	3.5	19	9.5	15.5
Aspiration	Nil	-	Nil	-	Nil	-	16	8	8
Renal Failure	Nil	-	Nil	-	Nil	-	3	1.5	1.5
External Injury	Nil	-	Nil	-	Nil	-	15	7.5	7.5
PROM	19	9.5	7	3.5	11	5.5	9	4.5	13.5
PPROM	Nil	-	Nil	-	Nil	-	4	2	2

In normotensive control 45pts (22.5%) suffering from anemia. 7(3.5%) were hypoproteinemic. No ante partum haemorrhage, 1(0.5%) post partum haemorrhage, 7 (3.5%) mal presentation (all are breech) 5 (2.5%) mal position, no urinary tract infection, no Diabetes, 1 (0.5%) twin pregnancy, 20 elevated liver function test, no HELLP syndrome, no fever, no aspiration, renal failure or external injury, where as 19 (4.5%) developed premature rupture of membranes but no PPROM (preterm PROM).

In mild PIH (92cases) – 14 (7%) were anemic, 6 cases (3%) were hypoproteinemic, no APH or PPH 5 cases (2.5%) of mal presentation, 3 (1.5%) cases of malposition, 6 (3%) had UTI 4 (2%) had associated gestational diabetes, no multiple pregnancies, no abnormality in LFT no HELLP syndrome, no aspiration, severe renal failure or external injury. 5 cases (2.5%) had fever, 7 cases (3.5%) developed PROM no PPROM.

In severe PIH (68cases) 9 (4.5%) were anemic 9 (4.5%) hypoproteinemic 3(1.5%) developed APH, all 3 are abruption grade I, one (0.5%) case of severe atonic PPH. 2 (1%) malpresentation. 2(1%) malposition, 18 (9%) developed UTI mostly catheter induced, associated GDM 2 (1%), no multiple pregnancy, 43 (21.5%) had abnormal LFT without HELLP syndrome, none developed HELLP,

fever in 7 (3.5%) no aspiration, severe renal failure, external injury. PROM in 11 cases (5.5%), but no PPROM.

In Eclampsia (40cases) 25 (12.5%) were anemic 24 (12%) were hypoproteinemic, 1 (0.5%) developed APH (abruption grade II), PPH in 1 (0.5%) malpresentation in 2(1%) cases, malposition in 3 (1.5%) cases, 23 (11.5%) had UTI, GDM -1case (0.5%) no multiple pregnancy, abnormal liver function test without overt HELLP syndrome in 37 (18.5%) cases, HELLP syndrome in 3 (1.5%) cases, 19 pts (9.5%) developed fever , 16 (8%) had aspiration, 3 patients (1.5%) developed severe renal failure, 15 (7.5%) had external injury due to fits, PROM in 9 (4.5%) cases, PPROM in 4 (2%) cases.

Table - 9

CLINICAL FEATURES

Oedema in normotensive and PIH patients

Grading of Oedema	Normotensive control 200 cases		Mild PIH 92 patients		Severe PIH 68 patients		Eclampsia 40 pts	
	No. of Patients	%	No. of Patients	%	No. of Patients	%	No. of Patients	%
No Oedema	48	24	Nil	-	Nil	-	Nil	-
Grade I edema up to ankle	143	71.5	61	30.5	11	5.5	Nil	-
Grade II up to Shin	9	4.5	21	10.5	22	11	5	2.5
Grade III Abd wall edema	Nil	-	10	5	28	14	6	3
Grade IV (Anasarca)	Nil	-	Nil	-	7	3.5	29	14.5

In normotensive (control) patients 48 cases had no Oedema legs. Where as 143 (71.5%) cases had Oedema up to ankle (bilateral pitting pedal Oedema –Grade-I) 9 cases (4.5%) had grade II Oedema (Oedema up to shin). No one had grade III and IV Oedema.

In mild PIH of 92 patients, 61 (30.5%) had edema grade I, 21 (10.5%) had edema gradeII, 10 (5%) cases had edema grade III. No one had edema grade IV.

In severe PIH (total 68) 11 (5.5%) pts – grade I edema, 22 (11%) grade II. 28 cases (14%) grade III, 7 (3.5%) pts developed grade IV edema.

In eclamptic patients (40 cases) 5 (2.5%) had edema grade II, 6 (3%) grade III and majority 29 cases (14.5%) had edema grade IV.

Table - 10

Albuminuria

Grading	Normotensive 200 patients		Mild PIH 92 patients		Severe PIH 68 patients		Eclampsia 40 pts	
	No. of Patients	%	No. of Patients	%	No. of Patients	%	No. of Patients	%
Nil or faint trace	196	98	3	1.5	Nil	-	Nil	-
1+	4	2	64	32	21	10.5	1	0.5
2+	Nil	-	25	12.5	28	14	10	5
3+ & above	Nil	-	Nil	-	19	9.5	29	14.5

4 (2%) patients out of 200 normotensive (control) group had Albuminuria.

In mild PIH (92cases) – 64 patients (32%) had 1+, and 25 patients (12.5%) had 2+ Albuminuria.

In severe PIH (68cases), 21 pts (10.5%) had 1+ & 28 patients (14%) had 2+ & 19 patients (9.5%) had 3+ and above albuminuria. In Eclampsia (40cases), 1 pt (0.5%) had 1+ & 10 patients (5%) had 2+ & 29 patients (14.5%) had 3+ and above albuminuria In these 200 PIH patients 57 patients (28.5%) had 24 hrs urinary protein > 300mg/L . Urine Specific gravidity range 1012 to 1014.

HYPERTENSION

Total of 200 PIH patients 92 patients (46%) had diastolic BP > 90 but <110 mm Hg and classified under mild PIH. Rest of the 108 (54%) patients are severe PIH patients i.e. their diastolic BP is >110mmHg.Of these 40 (20%) patients developed eclamptic fits.

Table - 11

Fundus examination in PIH Patients

Fundus Changes	No. of PIH Patients	%
Normal Fundus	130	65
Grade I retinopathy	61	30.5
Grade II	9	4.5
Grade III	Nil	-
Grade IV	Nil	-

In 200 total PIH patients 130 cases (65%) were found to be normal during Fundus examination. Prevalence of grade I retinopathy in 61 patients (30.5%) & grade II retinopathy in 9 cases (4.5%). No pts had grade III or IV changes.

Table - 12

Biochemical Changes

Bio chemical parameters	Mild PIH 92 patients		Severe PIH 68 patients		Eclampsia 40 pts		Control Group	
	No. of Patients	%	No. of Patients	%	No. of Patients	%	No. of Patients	%
Blood urea > 40mg / dl	Nil	-	22	11	25	12.5	Nil	-
Blood UricAcid > 4.5 mg/dl	23	11.5	54	27	33	16.5	Nil	-
Sr. Creatinine > 1mg%	Nil	Nil	38	19	34	17	Nil	-
Sr.Fibrinogen < 200 mg/ dl	9	4.5	46	23	31	15.5	Nil	-
Hb % Mild 8.5 gm to 11 gm%	48	24	29	14.5	18	9	45	22.5
moderate 6.5 to 8.4 gm%	3	1.5	5	2.5	19	9.5	Nil	-
Severe < 6.5gm %	Nil	-	1	0.5	3	1.5%	Nil	-
Serum bilirubin total > 1.2mg%	Nil	-	8	4	13	6.5	Nil	-
Serum total proteins < 6 gm	33	16.5	23	11.5	19	9.5	7	3.5
LFT rise	Nil	-	17	8.5	18	9	Nil	-
Platelet count < 1 Lk/cmm	Nil	-	Nil		5	2.5	Nil	-

40mg/dl is taken as cut off value for blood urea, no one were above this is mild PIH group. 22 cases (11%) of severe PIH and 25 patients (12.5%) of Eclampsia group were above this level. 23(11.5%) of mild PIH cases, 54 (27%) cases of severe PIH and 33 (16.5%) of cases of Eclampsia were having rise in serum uric acid level above 4.5% mg/dl.

38 (19%) of severe PIH and 34(17%) of Eclampsia patients, had rise in serum creatinine more than1mgs%

9 cases (4.5%) of mild PIH and 46 (23%) of severe PIH & 31 (15.5%) of Eclampsia pts were having serum fibrinogen level less than 200mg /dl. In mild PIH patient 48(24%) were mildly anemic, 3 patients (1.5%) were moderately anemic. In severe PIH 29 (14.5%) were mildly anemic 5 (2.5%) were moderately anemic 1 patient (0.5%) was severely anemic. In Eclampsia18 (9%) were mild, 19(9.5%) were moderate and 3 (1.5%) patients were severely anemic.

In mild PIH nobody had serum bilirubin > 1.2mg%

In severe PIH 8 cases (4%) had total bilirubin more than 1.2 mg % where as it was 13 (6.5%) in eclamptic patients.

In mild PIH 33 cases (16.5%) had less than 6 Gms total proteins, where as it were 23 (11.5%) patients in severe PIH and 19 (9.5%) cases in eclamptic group.

Rise in liver function test is not seen in mild PIH group, but it was present in 17 case (8.5%) in severe PIH group and 18 (9%) cases in eclamptic group.

Platelet count less than 1 lakh / cmm (thrombocytopenia) was seen only in eclamptic group of about 5 cases (2.5%)

In control group, all other parameters were normal except 45 cases (22.5%) of mild anemia & 7 (3.5%) of Hypoproteinemia..

In both control group and PIH group the electrolyte (Na^+, K^+ Cl^- HCO) were normal.

Table - 13

Mode of Delivery

Mode of Delivery	Mild PIH 92 patients		Severe PIH 68 patients		Eclampsia 40 pts		Normotensive Control	
	No. of Patients	%	No. of Patients	%	No. of Patients	%	No. of Patients	%
Labour Natural	58	29	38	19	14	7	104	52
LMC Forceps	6	3	5	2.5	Nil	-	17	8.5
Outlet Forceps	7	3.5	6	3.5	8	4	32	16
Vacuum	Nil	-	Nil	-	Nil	-	4	2
LSCS	21	10.5	19	9.5	18	9	43	21.5

In 200 normotensive control 104 (52%) delivered labour natural, 17 (8.5%) by Low midcavity Forceps delivery, 32(16%) delivered by outlet forceps and 4 (2%) cases delivered by vacuum. Lower segment caesarean section in 43(21.5%) cases

In mild PIH group 58(29%) Labour natural, 6(3%) LMC forceps delivery, 7 cases (3.5%) delivered by outlet forceps, No vacuum, 21 pts (10.5%) had LSCS. In severe PIH group 38(19%) delivered by labour natural 5(2.5%) delivery LMC forceps, 6 cases (3.5%) delivery by outlet, no vacuum, 19 cases (9.5%) delivered by LSCS.

In Eclampsia labour natural is 14 (7%), No LMC forceps delivery, no vacuum delivery, 8 cases (4%) deliver by outlet forceps and LSCS for 18(9%) cases.

TABLE – 14

Gestational age at delivery

Gestational age at the time of delivery	Control group		PIH group	
	No.	%	No.	%
> 37 weeks	182	91	117	58.5
34 to 37 weeks	18	9	44	22
28-34 weeks	-	-	21	10.5
<28 weeks	-	-	18	9

In normotensive control majority 182 cases (91%) delivered above 37 completed weeks. Few cases – 18 (9%) delivered before 37 weeks but after 34 weeks.

In PIH Patients 117 cases (58.5%) delivered at the gestational age above 37 completed weeks. 44 cases (22%) delivered between 34-37 weeks. 21 cases (10.5%) delivered between 28 to 34 weeks, and 18 cases (9%) delivered before 28 weeks.

Table - 15

Indications for LSCS

	Indications in PIH patient		In control	
	No. of Patients	**%**	**No. of Patients**	**%**
Fetal distress	26	14	12	6
Failed induction	20	10	11	5.5
Mal presentation (primi breech)	3	1.5	7	3.5
Previous LSCS with CPD	5	2.5	13	6.5
Uncontrolled APE	2	1	nil	-

Of the total no of 58 LSCS patients in PIH group, 28 cases (14%) were indicated for fetal distress, 20 (10%) for failed induction, 3 case (1.5%) were primi with breech presentation. Previous LSCS with CPD as an indication for repeat LSCS in 5 cases (2.5%), uncontrolled Ante partum Eclampsia in 2 cases 1%.

In control group of the total 43 LSCS patients fetal distress as a indication for 12 cases (6%) Failed induction in 11 cases (5.5%) malpresentation in 7 (3.5%) cases; previous LSCS with CPD in 13(6.5%) cases.

Table - 16

Type of Induction / Acceleration of Labour

Type of Induction / Acceleration	Normotensive 200		Mild PIH 92 patients		Severe PIH 68 patients		Eclampsia 40 pts	
	No. of Patients	%	No. of Patients	%	No. of Patients	%	No. of Patients	%
Spontaneous onset	88	44	26	13.5	16	8	7	3.5
ARM / Synto for acceleration	58	29	12	6	13	6.5	7	3.5
PG induction	37	18.5	49	24.5	39	19.5	23	11.5
ElectiveLSCS	17	9.5	5	2.5	Nil	-	3	1.5

P =0.000 X^2 =55.43 Statistically SIGNIFICANT

In control group spontaneous onset of labour in 88 (44%) cases, artificial rupture of membrane / oxytocin for augmentation of labour in 58 cases (29%), PG induction in 37 (18.5%) cases. Elective LSCS-17cases (8.5%).

In mild PIH group spontaneous onset of labour in 26 (13.5%) cases, ARM/ oxytocin augmentation in 12 (6%) cases, PG induction in 49 cases (24.5%) Elective LSCS –5 pts (2.5%)

In severe PIH group spontaneous onset of labour in 16 cases (8%), ARM / Oxytocin augmentation in 13 cases (6.5%), PG induction in 39 (19.5%) cases. No Elective LSCS.

In Eclampsia group spontaneous onset of labour only in 7 cases (3.5%), ARM /oxytocin augmentation in 7 (3.5%), cases, PGE2 gel/PGF2α tablet induction in 23 cases (11.5%). Elective LSCS-3cases (1.5%)

Table - 17

One minute Apgar of the baby in Normotensive patients and PIH

Apgar	PIH		Control	
	No. of Patients	%	No. of Patients	%
1/10 to 4/10	16	8	8	4
5/10 to 6/10	18	9	17	8.5
≥ 7/10	133	66.5	168	84
Dead born / still born	33	16.5	7	3.5
Early Neonatal death	16	8	14	7

P =0.00002934 X^2 =23.67 Statistically SIGNIFICANT

In control group, the one minute Apgar was≥ 7/10 in 168 cases (84%). In 17 cases (8.5%) it is 5/10 to 6/10. In 8 cases (4%) it is 4/10 to 1/10.

In PIH patients 133 cases (66.5%) had one minute Apgar more than 7/10. In 18 cases (9%) it is 5/10 to 7/10. In 16 cases (8%) one minute Apgar was between 1/10 to 4/10.

Total number of dead born (or) Stillborn in control group is 7(3.5%).

In PIH group it is 33 (16.5%). Early neonatal death in control group was 14(7%). In PIH group it was 16 (8%).

Table - 18

Sex Incidence of babies

Group	Male babies		Female babies	
	No. of Patients	%	No. of Patients	%
Control	108	54	92	46
PIH	103	51.5	97	48.5

P =0.61 X^2 =0.25 Statistically NOT SIGNIFICANT

In control group 108 (54%) delivered male babies. 92 (46%) delivered female babies. In PIH group 103 (51.5%) delivered male babies, 97(48.5%) delivered female babies.

Table - 19

Weight of the baby

Weight	Control Group		PIH	
	No. of Patients	%	No. of Patients	%
≥ 3.5 kg	11	5.5	9	4.5
3 to 3.4 kg	66	33	45	22.5
2.5 to 2.9 kg	102	51	64	32
2 to 2.4 kg	21	10.5	39	19.5
<2kg	nil	-	43	21.5

P =0.000 X^2 =49 Statistically SIGNIFICANT

In Control group 102 babies (51%) were between 2.5 to 2.9 kg, 66 (33%) were between 3 to 3.4 kg, 21 babies (10.5%) were between 2 to 2.4 Kg, 11 babies (5.5%) were equal (or) above 3.5 Kg, no baby was less than 2 Kg.

In PIH group 64 babies (32%) were between 2.5 to 2.9 kg, 45 babies (22.5%) were between 3 to 3.4 Kg, 39 babies (19.5%) were between 2 to 2.4 Kg, 9 babies (4.5%) were equal (or) above 3.5 kg, 43 babies (21.5%) were less than 2Kg.

Number of preterm babies in PIH group (<2 kg) were 43 (21.5%) none in control group.

Table - 20

Kick count valuation (Average) 12 hrs

Period of gestation in weeks	Primi		Multi	
	PIH	Normotensive	PIH	Normotensive
21 to 30 weeks	64	66	77	82
31 to 36 weeks	70	70	78	82
37 week & above	36	63	75	80

Table shows that DFMC in both primi & multi Para had been similar in both normotensive & PIH patients.

Table – 21

Average BPD

Period of gestation	BPD Measurement	
	Normotensive	PIH
22 weeks	5.9 cm	5.8 cm
24 weeks	6.2 cm	6 cm
26 weeks	6.8 cm	6.6 cm
28 weeks	7.4 cm	7.3 cm
32 weeks	8.2 cm	8.1 cm
36 weeks	8.9 cm	8.6 cm
38 + 3 weeks	9.3 cm	9.2 cm

The average B.P.D. values in both normotensive and PIH were almost nearer. BPD values in Hypertensive cases were little less; but negligible.

Table -22

Growth Rate Of BPD In Control & PIH Patients

Duration of pregnancy in weeks	Normal	Mild PIH	SeverePIH & Eclampsia
21 to 32 weeks	2.8 mm/wk	2.7 mm/wk	2.5 mm/wk
33 to 40 weeks	1.8 mm/wk	11.7 mm/wk	1.5 mm/wk

The BPD growth rate in mild PIH is similar with normotensive cases. But there is a definite decrease in growth of BPD in severe PIH and Eclampsia patient.

Table -23

FEMUR LENGTH IN NORMOTENSIVE/ HYPERTENSIVE PATIENTS

Duration in weeks	FL in cm		
	Normotensive	Mild PIH	Severe PIH/Eclampsia
22 weeks	3.9 cm	3.9	3.8 cm
24 weeks	4.2 cm	4.2	4.2 cm
26 weeks	4.9 cm	4.8	4.8 cm
29 weeks	5.6 cm	5.5	5.5 cm
32 weeks	6.3 cm	6.2	6.1 cm
35 weeks	6.9 cm	6.8 cm	6.7 cm
38 + 3 cm	7.5 cm	7.4 cm	7.2 cm

The femur length in different weeks of pregnancy in both normotensive & Hypertensive (Mild PIH, Severe PIH and Eclampsia) were similar.

Drugs used in Hypertensive disorders in pregnancy.

1. Sedatives- Pethidine, Diazepam, promethazine
2. Antihypertensive – Tab.Alphamethyldopa, Tab.Nifedipine, Sublingual Nifedipine.
3. Anti convulsant-Magnesium sulphate, phenytoin.

DISCUSSION

According to ACOG incidence of preeclampsia -5 to 8%.**Incidence of preeclampsia in KMC is 11%.**Considering the age groups, the maximum number of patients were found between the ages of the 21-25 years, 44.5% and 42% in normotensive group& in PIH complicating pregnancy respectively. This shows that in this study the child bearing period is earlier when compared to advanced countries where number of elderly primigravidae are encountered. **Young pts <20&>30yrs said to have increased incidence of preeclampsia(AMJ of O&G 1993).In 10 years, 1958-1967, in Aberdeen city, the incidence of severe pre-Eclampsia in first pregnancy was 4.8% varying from 4.2% in 15-19 years age group to 6.5% in those of 30 years of age and more.**

Studying the gravidity and parity in the normotensive group &, pre-Eclampsia in 200 cases, it is found that mild, severe pre-Eclampsia and Eclampsia occurred in primi Gravida more than the others.(Mild-29% Severe-18% Eclampsia-13%,Total=60%) as studies in literature. **Majority of cases say 75 to 80% occur during 1st pregnancy.(Clinical Obstetrics& Gynecology journal volume 42)**. **In Aberdeen city study by Nelson (1955) showed that 75% of all cases of pre-Eclampsia occurred in 1st pregnancy.**

In our series of 200 cases, there were 29 cases (14.5%) with recurrent pre-Eclampsia. **Sibai et al (37) showed risk of developing pre-Eclampsia in next pregnancy being 45.5%. Out of them 21%go for severe pre-Eclampsia.** On analyzing the number of cases who were registered and unregistered, among the 92 Mild preeclampsia 22 cases (11%) were booked while rest unbooked. In 68 severe pre-Eclampsia patients 52pts (26%) were unbooked and rest were booked. Among the 40 cases of Eclampsia 34cases (17%) were unbooked. In control group 78cases (39%) were unbooked. **The high percentage of unbooked cases in preeclampsia indicates that these patients attended the hospital only when they have some complaints like edema feet, scanty micturition or giddiness and found to have increase of blood pressure.**

On analysis the incidence of pre-Eclampsia, Eclampsia in relation to social class Table-5 shows that majority of our patients belong to the lower socio-economic strata, where there is poor nutrition and associated with conditions like anemia and Hypoproteinemia.

Considering the clinical aspects of hypertensive disorders, the three important clinical features – hypertension, edema and Proteinuria are changes of which the pregnant women are unaware except edema. By the time she has developed symptoms and signs that she herself can detect such as headache, visual disturbance, puffiness of the face and fingers, the disorder is usually advanced.

Among the various clinical features noted in 200 cases of hypertensive disorders complicating pregnancy the obvious gross finding was edema. There is a problem in determining what constitutes a normal pregnancy particularly with regard to clinical edema. Normality can be accepted in terms of absence of any obvious complications or any changes which has been shown to be associated with maternal morbidity or mortality. But this criteria **hypertension along with Proteinuria can be seen to be abnormal since it is associated with increased fetal loss (Nelson 1955).** Clinical edema alone on the other hand has not been clearly shown to be associated

with increased morbidity or mortality and must, therefore probably be considered as physiological.

Our study shows that edema increases with hypertension and when it is associated with anemia, hypoproteinemia, it took a longer time to clear up. **There was more number of anemic patients in preeclampsia. The most cardinal sign is hypertension.** Blood pressure was checked every hour during labour and 4 hourly after delivery. Blood pressure was recorded 48hrs, 72hrs, 1wk, 4wks&6wksafterdelivery.

ALBUMINURIA IN HYPERTENSIVE DISORDERS COMPLICATING PREGNANCY

Proteinuria varies greatly not only from individual to individual but also in the same women from time to time. The variability points to a functional (Vaso-spasm) rather than an organic cause. In early preeclampsia, Proteinuria may be minimal or entirely lacking. In more severe grades, proteinuria is usually demonstrable and may be as much 10gm/L. Proteinuria almost always develop later than the hypertension and usually later than excessive weight gain.

In our study mild PIH cases showed either traces or mild albuminuria. The moderate and heavy proteinuria was found in

severe PIH and Eclampsia cases thereby showing that the incidence of proteinuria increases with severity of PIH.

MATERNAL COMPLICATIONS vise

Study	APH	HELLP/DIC	RENAL FAILURE
Hall&colleagues2000	20%	5%	0.3%
Vissur&Wallenburg1995	5%	--	--
MurphyDJ & StirratGM 2000(22)	1.5%	21%	1.3%
OlahKS,Redman CW,Ge eH1993	--	14.2%	1.1%
Our study	2%	1.5%	1.5%

Our study showed relatively low incidence of APH & HELLP than Halls study. **Renal failure correlating with Murphy's study.**

The most common complication during pregnancy in 200 cases of hypertensive disorder complicating pregnancy was anemia.

MANAGEMENT

When Eclampsia develops ante partum or Intra partum the outlook for Mother and baby is worse when the delivery is delayed as Menon 1961 and HopesLera 1967 have shown in this series

Time for convulsion up to delivery	**Maternal Mortality**
0-2 hrs	7
2-4 hrs	13
4-8hrs	19
8-12hrs	25
12-24hrs	32
24hrs	42

Delivery should therefore be promptly effected by whatever means as this carries best prognosis for mother and baby. **Menon (1961) showed caesarean section to be safe when performed at the right time, when he reported loss of only one case of 42 eclamptic delivered by caesarean section during a 5 years period.**

Crichton, Notelovitz and Heller (19678) demonstrated improvement in perinatal mortality by early caesarian section of antepartum eclamptics. In 240 cases, delivered abdominally, perinatal mortality was 35% compared with 47% in 83 cases delivered

vaginally, the maternal mortality being equal. The improved prospects for babies weighing between 1360-2768gm. **Lopex Llera (1967) also reported improved fetal results from caesarian section**.

However it would seem reasonable to attempt vaginal delivery by inducing labour and monitoring the foetus carefully. Whatever method of delivery is chosen, avoidance of haemorrhage and operative shock are important.

Due to their pre existing hypovolemia and haemoconcentration, any blood loss is badly tolerated. Blood loss greater that 250 ml should be replaced by transfusion immediately irrespective of pre-operative hemoglobin level.

DRUGS

Drugs used in the treatment of hypertensive disorders complicating pregnancy in mild and severe variety of preeclampsia were mainly sedatives and anti hypertensive.

The sedatives used were Pethidine hydrochloride, Diazepam, promethazine, chlorpromazine , phenytoin, magnesium sulphate.

Mannitol was given in doses of 175ml intravenously in Eclampsia to reduce the cerebral edema. Anti hyper tensive used

wereT.Nifedipine, T.Alpha methyl dopa, Magnesium sulphate (50%).PRITCHARD regime followed.

The new born were examined soon after birth and Apgar at 1 mt noted. In Eclampsia&severe PIH, as described in literature the babies are in an unfavorable uterine environment in the later weeks of pregnancy and the intrauterine hypoxia is greater when compared with normotensive pregnancy and the distress increases as the severity of the disease increases.

WEIGHT OF THE NEW BORN

In our study The birth weight was more in the mild variety than the severe PIH, hence the fetal prognosis in mild variety is better.

SEX INCIDENCE OF NEW BORN

In our series of 200 cases, of hypertensive disorders complicating pregnancy, 51.5% of the new born were male, 48.5% were female. **The incidence of PIH seems to be associated with carrying a male foetus, as reported byButtner (1903), Hammershalf (1904), Kushner (1931), Salzmann (1955), Chesley (1978) and a study conducted in Aberdeen City between 1967-1979 and Doris M. Campbell andlan Mc Gillivary 1983.**

Author	No. of Pregnancies	Male	Female	Sex Ratio
Buttner 1903	1782	966	816	1.18

Hammershalf 1904	297	161	136	1.18
Kushner 1931	2013	1155	850	1.35
Salzmann 1955	421	222	199	1.12
Chesley 1978	274	128	146	0.88

Sex ratio at birth is No. of males born for every 100 female.

Dr. Shirin Mehtaji has stated in her series of study in Camas and Albles Hospital, Bombay, that the incidence of perinatal mortality due to hypertensive disorders complicating pregnancy being 15.63/ 1000 total births. In our study **the perinatal mortality rate due to PIH is 26.2/ 1000 total births.**

Among the normotensive patient monitored the outcome for both the mother and foetus is satisfactory. In normotensive patient rate of growth of BPD was 2.8 mm/wk up to 32 wks, and 1.8 mm/wk from 33 wks onwards. The fetal birth weight within normal curve. All cases showed positive non- stress test. The fetal birth weight in these patients is majority in the range of 2.5to3kg.

Of the 156 cases of unbooked hypertensive pregnancies 70 were mild Preeclampsia 52, were severe Preeclampsia, 34 were Eclampsia. Regarding maternal fetal complications unbooked cases were having more complications than booked one.

Complications	Total Cases	Unbooked PIH	Booked PIH

Anemia	48	41	
Hypoproteinemia	39	32	7
APH	4	3	7
PPH	2	2	-
Malpresentation	9	8	1
Malposition	8	6	2
UTI	47	43	4
DM	7	6	1
Abnormal LFT	80	63	17
HELLP	3	3	-
Fever	31	27	4
Aspiration	16	13	3
Renal failure	3	3	--
Ext. Injury	15	12	3
PROM	27	23	4
PPROM	4	3	1

Of the total 48 cases of anemia 41 were unbooked. 3 APH out of 4, all 2 PPH, 8 out of 9 malpresentation and 6 out of 8 malposition were unbooked cases. 43 out of 47 cases of UTI, 6 DM, 63 abnormal LFT, 27 fever cases, 13 aspiration cases, 12 external injury were seen in unbooked patients. All 3 cases who developed HELLP syndrome died were unbooked. Of the 27 PROM and 4 PPROM cases 23 PROM and 3 PPROM were unbooked.

Regarding fetal complications of the total 33 dead born/still born babies 27 were unbooked and in 16 cases of neonatal death 10 were unbooked.

This study clearly indicates the importance of booking in terms of maternal, fetal morbidity and mortality.

The few frequent complications were Eclampsia, IUGR, and ante partum haemorrhage and intra uterine death. In the monitoring and regular follow up in the 200cases of PIH many aspects were analyzed.

This disorder was seen more frequently in primigravida than in multigravidea. Even in primigravidae very young primi (age group 16-19 years) and elderly primi (age group 30-34 years) were very commonly affected. In this group taken for analyses, 92pts were of milder variety and 68pts were of severe variety. This shows that the disease usually takes a milder form at the onset in majority of cases, when hypertension and proteinuria were definite criteria of the disorder. Cases especially mild hypertensive types were presenting without edema. But at the same time edema had been one of the triad of symptoms in severe PIH cases.

All patient of PIH were admitted in the hospital for investigations. Mild cases responded well with bed rest, sedatives, anti hypertensive (if needed) improved nutrition, and were then treated as outpatient with a weekly antenatal checkup. Severe cases needed admission for a longer duration.

The kick count maintained by both primigravidae and multigravidea. The USG examinations of the various fetal parameters done at 3-4 weeks interval showed an almost normal growth rate of biparietal diameter, femur length in mild preeclampsia.

The growth rate though very slightly lesser in milder hypertensive women, it was negligible. But at the same time, the growth rate of different parameters in severely hypertensive women were notably lesser than the normal pregnancies. The BPD growth rate in mildly hypertensive women was 2.8 mm/wk from 24-32 wks and 1.88 mm/wk from 32 wks onwards. The similar measurement in severely hypertensive women was 2.5 mm/wk from 24-32 wks and 1.5 mm/wk from 32 wks onwards.

REGARDING INDUCTION WITH PROSTAGLANDIN

Indications for PG induction

PG Induction in Eclampsia – 23 cases

PG induction in severe PIH - 39 cases

PG induction in mild PIH - 49 cases

Those who had Eclampsia induction was done for maternal goodness in 19 cases. 4 cases induced for PPROM before 34 weeks.

Of the 39 cases of severe Preeclampsia induced with PG 20 cases were induced for fluctuating BP, not properly controlled with anti-hypertensive drugs. 9 cases were induced for 37 completed weeks (term). Remaining 10 cases were induced for PROM.

Of the 49 cases of mild Preeclampsia induced with PG-7 cases for PROM, 42 for term.

REGARDING THREE MATERNAL DEATHS

All three maternal deaths were unbooked cases

- Two Primi – one 18 yrs old & one 36 yrs old and one G_2 P_1 L_1 24 yrs old.
- All three are antepartum eclampsia, developed HELLP syndrome and succumb to death in spite of termination of pregnancy and aggressive supportive treatment for the condition.

SUMMARY

Incidence of preeclampsia in KMC is **11%.** In our study there were 200 patients of mild, severe preeclampsia and Eclampsia were compared with 200 normotensive patients in terms of maternal and perinatal out come.

- **60%** of the preeclampsia Eclampsia patients were primi.
- **42%** were in the age group 21-25 years.
- **78%** were un booked
- **98%**of them belong to class 4or 5 socio economic status.
- **5.5%** had history of previous abortions and / or IUD.
- **14.5%**were recurrent PIH
- About **9.5%** had history of Hypertension in the family.
- Complications like anemia, hypoproteinemia, antepartum Haemorrhage, post partum Haemorrhage, urinary infection and abnormal liver function test, fever, aspiration, severe renal failure, HELLP syndrome, and external injury due to fits are more in patients with PIH mainly severe PIH and Eclampsia.
- **3** Patients developed HELLP syndrome, and all **3** died.
- Clinical features like grading of Oedema and albuminuria were in ascending order with severity of PIH.
- **65%** of PIH patients were having normal Fundus examination but **30.5%** had grade I retinopathy and **4.5%** had grade II retinopathy.
- Regarding biochemical parameters- With severity of PIH there were ascending order of biochemical abnormality.

- **71%** of them delivered via naturalis
- LSCS was most commonly indicated for fetal distress **(14%)**
- **55.5%** of PIH patients particularly severe variety and Eclampsia were induced with prostaglandin.
- **58.5%** delivered at term
- **4%** taken for elective LSCS.
- **66.5%**of babies born to PIH mother were normal with good Apgar (≥7/10)
- **33** babies were dead born/still born.
- **16** babies died in the early neonatal period.
- There was a slight male preponderance among babies (**51.5%**)
- **32%**of the babies weighed between 2.5 to 2.9 kg.
- Total number of maternal death among PIH group is **3**. All three died due to **HELLP** syndrome
- Maternal mortality due to pregnancy induced hypertension is **3 (1.5%) in 200 cases of PIH**.
- Perinatal mortality rate = No of **still born** + **neonatal death** / 1000 **live birth**.
- In our study **the perinatal mortality rate due to PIH is 26.2/1000.**

CONCLUSION

This is the random study of 200 cases of maternal and perinatal outcome in mild and severe pre Eclampsia and Eclampsia in comparison to the outcome in 200 normal gravidae.

Even though it is a random study it shows that the occurrence of preeclampsia and Eclampsia are found mostly in unbooked cases.

There is an increase in incidence of maternal and perinatal morbidity and mortality in PIH patients, particularly with severe preeclampsia and Eclampsia. This study clearly illustrate that the regular monitoring of antenatal mothers with hypertension during pregnancy and labour has a welcoming result in modern obstetrics. Careful antenatal monitoring helps to identify the early warning signs of Eclampsia. Prompt care at this stage is often successful in preventing the high morbidity and mortality. The need for monitoring the pregnancy for early detection and proper management of a complication of pregnancy, with timely intervention as regards termination of pregnancy with facility for monitoring during labour and after care of the high risk infant, seems to play more important role in the final outcome.

Both the foetus and maternal factors are to be taken into consideration. Once the blood pressure in these cases settles down with bed rest, sedatives and antihypertensive drugs, fetal factors are to be considered and decision taken accordingly to whether or not the foetus is in difficulty or whether it might benefit from continued intra-uterine maintenance. If tests for fetal well being are normal and provided the mother's condition does not threaten life, the pregnancy is allowed to continue until fetal maturity is reached.

Once the fetal maturity is reached and confirmed by the various tests, termination of pregnancy is to be considered. Mode of delivery should be decided accordingly. This regimen of monitoring of PIH will definitely improve the pregnancy outcome.

and

More awareness to be created regarding early and regular antenatal check up to improve the pregnancy outcome in PIH patients.

ABBREVATION

1.	ACOG	- American Journal of Obstetrics and Gynaecology
2.	AST	- Aspartate Transaminase
3.	ALT	- Alanine Transaminase
4.	AFI	- Amniotic Fluid Index
5.	A	- Alive
6.	AP	- Antepartum
7.	BJOG	- British Journal of Obstetrics and Gynaecology
8.	BT	- Bleeding Time
9.	BP	- Blood Pressure
10.	BMI	- Body Mass Index
11.	CT	- Clotting time
12.	DIC	- Disseminated Intravascular Coagulation
13.	DM	- Diabetes Mellitus
14.	EL	- Eclampsia
15.	FD	- Fetal Distress
16.	GA	- Gestational Age
17.	HCG	- Human Chorionic Gonadotrophin
18.	H_2O_2	- Hydrogen Peroxide
19.	IE	- Imminent Eclampsia
20.	IP	- Intrapartum
21.	ML	- Mild Pre Eclampsia
22.	ND	- Neonatal Death
23.	P	- Primi
24.	PN loss	- Perinatal Loss
25.	PP	- Postpartum
26.	PT	- Prothrombin Time
27.	PE	- Preeclampsia
28.	PIH	- Pregnancy Induced Hypertension
29.	PGI_2	- Prostacyclin
30.	NST	- Non Stress Test
31.	NICU	- Neonatal Intensive care unit
32.	SVR	- Severe pre Eclampsia
33.	T	- Total
34.	TG	- Triglyceride
35.	TXA_2	- Thromboxane A2
36.	USG	- Ultra sonogram
37.	VEGF	- Vasoactive endothelial growth factor

PROFORMA

1. SL. No. :
2. Name :
3. Age :
4. IP No :
5. Obstetric score :
6. Gestational age at admission :
7. Socio economic status :
8. Height :
9. Weight :
10. Booked / un booked :
11. H/O previous bad obstetric history

    Recurrent IUD/ Abortion :

12. Any imminent symptoms :

13. Investigation

    a. Hb% :

    b. Urine alb :

    Sugar :

    Deposit :

Bile salts, bile pigments :

c. Blood urea, sugar :

d. Sr. creatinine :

e. Sr. electrolytes $\frac{Na^{+} \mid K^{+}}{cl^{-} \mid H\ co3^{-}}$ **:**

f. Sr. Uric acid :

g. Sr. fibrinogen :

h. Complete haemogram including platelet count & peripheral smear. :

i. LiverFunctionTests

ALT :

AST :

Alkalinephosphatase :

BT :

CT :

j. Sr. Bilirubin- Total, Conjugate :

k. Albuminuria Nil/ 1+/ 2+/3+/ >3+:

I. 24 hrs urinary protein :

14. Present history :

15. Relevant past history- Medical history

surgical history :

16. Relevant personal History :

17. Menstrual history :

18. Marital History :

19. Obstetric history :

20. Complication (maternal) during

present pregnancy like anemia

Hypoproteinemia etc. :

I –Trimester :

II – Trimester :

III- Trimester :

21. Drug History – present, past :

22. Gravidogram – Date, urine albumin,

wt, SFH, AC, BP, imminent

symptoms. **:**

23. Magnesium sulphate chart **:**

Time, dose, PR, RR, BP, Temperature,

Urine output, knee-jerk. **:**

General examination **:**

PR **:**

BP **:**

Temperature **:**

CVS **:**

RS **:**

Anemia / Not **:**

Grading of edema- 0,1,2,3,4 **:**

P/A **:**

P/V **:**

25. Mode of Delivery Labour

natural / LSCS **:**

26. Mode of Induction/ acceleration :

27. Admission delivery interval :

28. If LSCS – Indication for LSCS :

BIBLIOGRAPHY

1. W.M. Romano; L.A. Lukash, B.Sc., Mitchell M.D: Substrate utilization for estrogen synthesis by human fetal membranes and deciduas- American Journal Obstetrics and Gynecology 1986, **155:** 1170-1175.

2. Frank O.Page: James N. Martin, sue M. Palmer; Rick W. Martin – Correlation of acid base status with Apgar scores, fetal heart rate tracing – American Journal of Obstetrics and Gynecology 1986, **154:** 1306-1311.

3. Bonner J. McNicol G. Douglas (1971) British Medical Journal ii, 200.

4. Gant N.F. Chand. S., Worley R.J., Macdonald P.c. (1974) American Journal of Obstetrics and Gynecology 120,1.

5. John P. Gudson Jr. M.D., Stephen G. Anderson, M.D. – Clinical evaluation of the “roll over test” for pregnancy induced hypertension – American Journal of Obstetrics and Gynecology volume 127, No.1 January 1, 1977.

6. MacGillvray 1958- Some observations on the incidence of pre – Eclampsia Journal of Obstetrics and Gynecology of British Commonwealth 65:

7. Fenny and Scott – Pre – eclampsia and changed paternity – Reproductive Biology, 1980.

8. Ikedife D (1980) – Eclampsia in multipara – British Medical Journal 89: 862-863.]

9. Chng 1982, Immunology of pre-eclampsia British Medical Journal 89: 862-863.

10. Chun et al, 1964. Clinical observation on some aspects of Hydatiform mole- Journal of obstetrics and Gynaecology of British Commonwealth 71.

11. Jeffcoate and Scott J.S. 1959- some observations of placental factor in pregnancy toxemia – American Journal of Obstetrics and Gynecology 72: 475-489.

12. Brosen I.A. RObertson; X Dixon H.G. 1972, Role of spiral arteries in the pathogenesis of pre eclampsia – Obstetric and Gynecology Annuals.

13. Campbell 1983; Griffin Dr. Diaz Recasen New Doppler Technique for assessing uteroplacental blood flow.

14. Green Wald p-1963- Chronic fetal distress and placental insufficiency – Biology of Neonates 5: 215-265.

15. Abdul Kareem 1961. The effect of pressor response in Preeclampsia. J. Obstetric Gynecology 82: 246, 1961.

16. American Journal of Obstetric & Gynecology 2002.

17. Bardeguez Ad, Mc Nerncy R, Frieri M, Verma UL, Tejani N. Cellular immunity in preeclampsia. Alterations in T-lynphocyte subpopulations duringearlypregnancy obstetric – Gynecology 773859, 1991.

18. Chang et al. Effect of endothelium – derived relaxing factor inhibition on the umbilical placental circulation in fetal lambs in utero.

19. Chang et al. Effect of endothelium – Derived relaxing factor inhibition on the umbilical placental circulation in fetal lambs in utero. AM J. Obstetric –Gynecology 166: 727, 1992.

20. Cunningham FGI, Lindheimer MD: hypertension in pregnancy. Current concepts. N. England. J. Med 326: 927: 972, 1992.

21. Dekker GA, Sibgai BM: Etiology and pathogenesis of preeclampsia current concepts AM J. Obstet – Gynacol 179: 1359-1998.

22. De BOer K, Buller HR, Ten cate JW, Treffers PE. Coagulation studies in the syndrome of HELLP BR, J. Obstet Gynacol 98: 42, 1991.

23. Haddad B, Louis – sylvestre C, Doridot V, Touboul C, abirached F, Paniel BJ. (Criteria of pregnancy termination in women with preeclampsia) Gynecol obstet Fertil. 2002 June; 30(6): 467-73. French.

24. Haddad B, Sibai BM. Expectant management of severe preeclampsia: proper candidates and pregnancy outcome. Clin Obstet Gynecol. 2005 Jun; 48(2): 430-40. Review.

25. Hall DR, Odendall HJ, Steyn DW, Grove D. Urinary protein excretion and expectant management of early onset, severe pre-eclampsia. Int J Gynaecol Obstet. 2002 Apr; 77(1):1-6.

26. Hall DR, Odendall HJ, Steyn DW. Expectant management of severe pre-eclampsia in the mid-trimester. Eur J Obstet Gynecol REprod Biol.2001 Jun; 9692):168-72.

27. Hall DR, Odendall HJ, Kirsten GF, Smith J, Grove D. Expectant management of early onset, severe pre-eclampsia: maternal outcome. BJOG. 2000 Oct; 107(10): 1252-7.

29. Hayman R, WArren A, Brockleysby, J. Johnson I, Baker P. Endothelial dysfunction in preeclampia. Br J Obstet Gyneol 107:108, 2000

30. Leduc L, Wheeler JM, Krishon B, Mitchell P, Cotton DB Coagulation Profile in severe preeclampsia Obstet – Gynacol 79:14, 1992.

31. Nassar AH, Adra AM, Chakhtoura N, Beyodouns. Sever preeclampsia remote from term. Labour induction or elective cesarean delivery? AM. J. Obstet gynaecol 1979: 1210, 1998.

32. North RA, Taylor RS, Schellenburg J.C. Evaluation of a definition of preeclampia. Br J Obstet Gynaecol 106:767, 1999.

33. Odendall HJ, Hall DR, Grove D Risk factors for and perinatal mortality of abruptio placenta in patients hospitalized for early onset severe pre-eclampsia – a case controlled study. J. Obstet Gynaeco. Jul; 20(4):358-64.

34. Odendall HJ, Steyn DW, Norman K, Kirsten GF, Smith J, Theron GB.Improved perinatal mortality rates in 1001 patients

with severe pre-eclampsia. S Afr Med J. 1995 Oct; 85(10 suppl):1071-6.

35. Olah KS, Redman CW, Gee H. Management of severe, early pre – eclampsia: is conservative management justified? Eur J Obstet Gynecol Reprod Biol. 1993 Oct 29;51(3):175-80.

36. Oosterhof H, Voorhoeve PG, Aarnoudse JG Enhancement of hepatic artery resistance to blood flow in preeclampsia and in HELLP. AM J. Obstet – Gynacol 171:526,1994.

37. Redman CWG, Sacks GP, Sargent K Preeclampsia an excessive maternal inflammatory response to pregnancy. AM J. Obstet Gynacol 180:499-1999.

38. Roberts JM: Preeclampia: what we know and what we do not know. seminar perinatoll 24:24, 2000.

40. Silver H, Morgan T, Ward K.Blood volume stratified by angiotensinogen genotypes in normal and hypertensive pregnancies. Presented at 21st annual meeting of society of maternal – fetal medicine –fetal medicine, February 5-10, 2001 held in Reno, Nerada.

41. Sibai BM. Diagnosis and management of gestational hypertension and preeclampsia. Obstet Gynecol. 2003 Jul; 10291):181-92. Review.

42. Sibai BM. Friedman SA, Schiff E, Lubarsky SL, Expectant management of severe preeclampsia remote from term. Clin Obstet Gynecol. 1999 Sep; 42(3):470-8. Review.

43. Visser W, Wallenburg HC. Maternal and perinatal outcome of temporizing management in 254 consecutive patients with severe pre- eclampsia remote from term. Eur J Obstet Gynecol Reprod Biol. 1995 Dec; 639(2): 147-54.

44. Sibai BM, Mercer BM, Schiff E, Friedman SA. Agreessive versus expectant management of severe preeclampsia at 28 to 32 weeks' gestation: a randomized controlled trial. Am J Obstet Gynecol.1994 Sep; 171(3):818-22.

45. Yang Z, Li R, Shi LY, Wang LN, Ye RH, Wang R, Huang P. (Clinical delimitation and expectant management of early onset of severe pre-eclampsia) Zhonghua Fu Chan Ke Za Zhi. 2005 May; 40(5):302-5. Chinese.

46. Golden Burg R.L., Huddleson J.F. Nelson K.G. Apgar Scores and umblical aroterial PH in Preteion New born infants –

American Journal Obstetrics and Gynaecology 1984, 149-651.

47. Arcilla Shadmi, Roy Homburg and Vaclar Inster – An examination of the relationship between fetal movements and infant motor activity – Acta obstetricia et Gynacologica Scandinavia Vol.65:1986, 335-339.

48. Lill Valentin, Karel Marral and Lars Wahlgren – Subjective recording of fetal movements – screening of a pregnant population, the clinical significance of decreased fetal movement counts – Acta obstericia et al Gynaecologica – Scandinavia Vol. 65:753-758, 1986

49. Sandovsky E. Yaffee H. Daily fetal movement recording and fetal prognosis obsetrics Gynaecology 1973, 41:845-850.

50. Wheeler. T., Gennser. G, Lindwall. R., Murritts A.J. Changes in the fetal heart rate associated with fetal breathing and fetal movements – British Journal Obstetrics and Gynaecology.

51. Boylan. P., Lewis. . P.J., Fetal breathing in Labor. Obstetrics Gynaecology 1980, 56: 35-38.

52. Raybur W.F., Mekean. H.E., Maternal perception of fetal movements and perinatal outcome – Obstetrics Gynaecology 1980, 56:161-164.

53. Hadlock F.P., Harrist R.B., Carpenter R.J. Sonographic estimation of fetal weight. Radiology- 1984, 150:535.

54. Chamberilin P.F., Manning F.A., Morrison, Harman C.R., Ultrasound evaluation of amniotic fluid volume 1. The significance of marginal and decreased amniotic fluid volumes to perinatal outcome. A.M.J. Obstetrics and Gynaecology 1984, 150:250.

55. Micael Y. Divon, Paul Chamberlin, Louis Sipos- Identification of the small for gestational age fetus with the use of gestational age – independent indices of fetal growth.

56. Devi, P.K. Menon, M.K.K. and Bhakar Rao, K., Study of foetal weight/placental weight ratio- post-graduate obstetrics and Gynaecology – 2nd edition 10: 1982.

57. Meera Agarwal, Manju Khemani, Kamal Buckshee – Acceleration Fetal Movement ratio and neonatal outcome – Journal of Obstetrics and Gynaecology of India.

58. Chisley K.c.- Hypertensive disorders or pregnancy p.227, 1978.

59. Sudha N. Tandon, Geetha S. Rao and Prathiba R. Vaidhya- Antenatal electronic foetal monitoring in High Risk Pregnancies – Journey of Obstetrics and Gynaecology of India, 868-871.

60. Raphael J.P., Kuhn non stress antenatal cardiotocograph in High Risk pregnancy- Asia Oceana Journal Obstetrics and Gynaecology, Vol.8, No.1 March 1982.

61. V.L. Bargava and M.s. Greval – Heriditary factor in Pregnancy induced hypertension Indian Journal of obstetrics and Gynaecology 84:277-281.

62. Norbart- Gleicher, M.D., Leo R. Boler – Hypertensive Disease in Pregnancies and parity – American Journal of obstetrics and Gynaecolgoy 1986, 154:1044-1049.

63. T.Y. Khong, W.B. Rolbbertson – Vascular response in adequate to placentation in pre-eclampsia and small for gestational age infants – British Journal of Obstetrics and Gynaecolgoy October 1986 volume 93: 1049-1059.

64. Campbell et al – Routine ultra sonar screening for prediction of gestational age – obstetricia and Gynaecologica 65:613.

65. Rosenberg 1982, Antenatal detection of IUGR- British Journal of Obstetrics and Gynaecology 89.

66. Fedrick and Adelstein 1978- Factor associated with low birth weight of infant delivered at term-British Journal. of obstetrics and Gynecology 85.1-7.

67. Westin B. 1977, Gravidogram and fetal growth – Octa obstetricia and Gyhnaecologicia Scandinavia 56:273-282.

68. Westin, Vest Neelson 1983, Oestriol screening in Pregnancy – Octa obstriticia and gynaecologica scandinavia 52:

69. Wheeler et al 1979, Computer Analysis of fetal heart rate during normal pregnancy-British Journal Obstetrics Gynecology 86.

70. Kubli 1984- Antenatal monitoring of PHR- fetal physiology and Medicine – Basis of Perinatalogy.